FURTHER EXPERIENCE WITH THE DICHLORAMIN TREATMENT OF MASTOID WOUNDS.*

BY RALPH A. FENTON, M. D.,
PORTLAND, ORE.

The advantages of chlorin-yielding solutions in wound dressings were thoroughly discussed during the World War, and extensive literature bearing on this topic is available. Surface irritation from aqueous solvents led to the use of various oily materials which released chlorin slowly in contact with wound surfaces, and among these one of the best was dichloramin-T, 2 per cent, dissolved in eucalyptol or in chlorcosane. Obvious disadvantages in retaining watery solutions within shallow bone cavities by two-hourly injection or constant flow suggested the use of dichloramin-T to us as a first dressing in mastoid wounds, in 1918. We have continued this use for eleven years, following the details of the technic published in our report of 1921 before the Pacific Coast Oto-Ophthalmological Society on forty-five cases. At that time we reported marked reduction in duration of postoperative temperature, rapid disappearance of purulent discharge from the wound, and shortening of stay in hospital.†

Recent inquiry on the Pacific Coast shows that but few otologists who started using this method in 1921 or 1922 have continued with it. Some state that they found no advantage over a “modified blood clot.” In general the discontinuance has been in the nature of a return to plain gauze packing or rubber tube drainage of the mastoid cavity, without chemical disinfection. Some surgeons stated that they disliked to bother with an additional detail at the end of such operations.

*From the Departments of Otolaryngology and Bacteriology, University of Oregon Medical School, Portland.
Careful examination of our records of several hundred cases done since our previous report bears out our earlier opinion that dichloramin-T is definitely inhibitory of bacterial growth in bone wounds. Contrary to the opinion of many, mere surgical scraping of bone is an insufficient guarantee against continuance of bacterial infestation. Macroscopically clean cavities will be found to give teeming cultures if a swab be taken just before suturing; and it is not possible to be sure that every Haversian canal is free from microbes. Of course, removal of gross lesions, necrotic trabeculae and purulent exudates, with free washing in of uncontaminated blood and the stimulus of trauma in fostering leucocytic activity, will in most cases aid individual resistance in bringing about prompt healing without the help of bactericides.

It is, however, not infrequently a matter of tragic moment that the surgeon, especially in streptococcic cases of the type recently prevalent, can never be sure that he has reached the limit of infected bone.

For this reason we have recently conducted, with the help of the Department of Bacteriology, a careful study of the actual effect of dichloramin-T in chlorcosane upon human-blood agar plates inoculated with fresh virulent cultures from a series of acute mastoid cases, all in hospital during March, 1929. All were chosen because of the severity of their infection with hemolytic streptococci.

The examination was made as follows: Cultures from a single bacterial colony were made in infusion broth. One drop from this 24-hour culture was placed on a blood agar plate and smeared with a glass rod. Three plates were made from each culture and numbered I, II, and III. Ten to twelve drops of dichloramin-T, 2 per cent in chlorcosane, was placed on each plate II and smeared over the whole surface with a glass rod. All three plates were then incubated at 37½° C. for 24 hours. An equal amount of DCT was then placed on each plate III, smeared over the surface and incubated an additional 24 hours.

With cultures A, B, and C, 1 cc. of sterile saline solution was then pipetted on to each plate and the surface rubbed with a glass rod, to wash off the colonies. One loopful of saline from each such plate was then inoculated into a poured blood agar plate, which was incubated 24 hours.

With cultures D, E and F, an area of about one square cm. was rubbed with a loop and subcultures made in blood agar without washing.

Cultures of F and G were made in infusion broth in three test tubes labeled I, II and III. Immediately after inoculation 1 cc. of DCT was poured into tube II. All three tubes were then incubated 24 hours. An equal amount of DCT was then poured into tube III, and the three tubes were reincubated 24 hours. Subcultures were then made by diluting in saline into blood agar plates.

Results were as follows:

<table>
<thead>
<tr>
<th>Case</th>
<th>Plate (no DCT)</th>
<th>Plate (DCT immediately)</th>
<th>Plate (DCT added 24 hrs. later)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. A</td>
<td>23 colonies</td>
<td>1 colony</td>
<td>1 colony</td>
</tr>
<tr>
<td>I. B</td>
<td>1700</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>I. C</td>
<td>1500</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>I. D</td>
<td>2500</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>I. E</td>
<td>1500</td>
<td>4</td>
<td>contaminated--no strep.</td>
</tr>
<tr>
<td>I. F</td>
<td>9000</td>
<td>10</td>
<td>1</td>
</tr>
</tbody>
</table>

Broth cultures:

<table>
<thead>
<tr>
<th>Tube I</th>
<th>Tube II</th>
<th>Tube III</th>
</tr>
</thead>
<tbody>
<tr>
<td>F.</td>
<td>380 million bacteria per cc.</td>
<td>840,000 per cc.</td>
</tr>
<tr>
<td>G.</td>
<td>468 million per cc.</td>
<td>11,520,000 per cc.</td>
</tr>
</tbody>
</table>

Other plates were inoculated and one-half was smeared with the oil. Inhibition of bacterial growth was strikingly evident in the covered half, but on account of the red medium could not be clearly photographed. Summarizing the results of the studies by plating, which roughly reproduce the effects of bacterial growth in a film of blood-clot overlying the fresh cut bone of the mastoid, we find reduction of colonies from one-twentieth to one-twenty-five-hundredth of the number multiplying without exposure to dichloramin. In the tubes of broth, which indicate roughly the speed of bacterial multiplication in serosanguineous exudates at body temperature, dichloramin reduces growth 400 times in one case and 40 times in another during 24 hours; while after 48 hours there is no growth at
all. The average proportion of reduction of growth from the plate cultures under dichloramin is one-nine-hundredth of the ordinary amount, in this series and for these strains of hemolytic streptococci from the mastoid.

Otherwise stated, we feel that our belief is justified in the inhibitory action of dichloramin-T upon bacterial growth in acute mastoid wounds; and we feel that herein lies the explanation of the clinical facts heretofore presented, namely, the rapid disappearance of pus from wounds so treated, formation of healthy granulations, slight toxic absorption, lessened superficial necrosis and tissue loss. The resultant wound exudate is characteristically mucoid, reddish and relatively low in exfoliated cellular structures. Recurrence of yellow pus in the ear, or in a wound so treated, is suggestive of re-infection of the ear and mastoid from the throat, or of extension of the purulent process into unopened cellular structures.

Dichloramin is useful daily during the first week or ten days of the healing process; thereafter mercurochrome or silver solutions are used externally, as may seem necessary to secure smooth healing.

Our preference is still for the wide open wound, with one or two sutures at the ends. Such a wound is filled full of the oil, which is “held in” by a lightly inserted fold of narrow oil soaked gauze packing. Removal and insertion of such oil soaked material is painless; the depths of the wound are not packed, swabbed or otherwise disturbed.

Conclusion: Bacteriologic examination based on virulent cultures bears out eleven years of clinical experience in many hundred cases, demonstrating the value of dichloramin-T, 2 per cent in chlorcosane, as a routine dressing in acute mastoid surgery, not only for its remarkable inhibitory action on bacterial growth but also for shortening materially the duration of after treatment.